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RELATIVE INSENSITIVITY OF AVIAN SKELETAL MUSCLE GLYCOGEN TO NUTRITIVE STATUS[1,2]

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Previous studies in avian species have reported time-dependent losses in muscle glycogen with prolonged feed withdrawal (FW). However, cervical dislocation was used to collect tissues, a method that results in significant involuntary muscle convulsions. In this study, cervical dislocation alone was found to reduce muscle glycogen by 23%, therefore, barbiturate overdose was used to collect tissue samples before and after FW, at the end of refeeding, and from continuously fed controls at each interval. Additionally, plasma samples from 6-wk-old male chickens were taken at the initiation and end of a 24-hr feed withdrawal, and at various times during refeeding. After 24 hr of FW, liver glycogen decreased markedly (77%; $P < 0.05$), whereas muscle glycogen decreased slightly and transiently, such that it returned to and remained at control levels, even after prolonged (72 hr) FW. Plasma glucose was decreased, whereas glucagon was elevated after a 24-hr feed withdrawal ($P < 0.05$), when compared with control concentrations. Muscle glycogen levels were not significantly increased over control levels after refeeding, but liver glycogen was increased by 380% ($P < 0.05$). Feed deprivation followed by refeeding resulted in increased circulating insulin and glucose levels when compared with control levels. Therefore, by using methods of tissue collection that ensure that muscle glycogen determinations are not confounded by artifactual degradation, these results verify that regulation of avian muscle glycogen stores is similar to that in mammals.

INTRODUCTION

Skeletal muscle glycogen is important in the chicken not only as a source of energy for contracting muscle, but also as an important component influencing the intracellular storage of water in muscle (1). Although feed deprivation and refeeding effects on both muscle and liver glycogen are well characterized in other species, the literature on poultry, which exhibit elevated plasma glucose concentrations and basal metabolism when compared with mammals, is limited and conflicting.

In mammalian species, glycogenolysis in muscle is largely a function of contraction-induced calcium release and activation of phosphorylase (EC 2.4.1.1) by catecholamines (12). Calcium released from the sarcoplasmic reticulum binds to the regulatory protein calmodulin, a component of phosphorylase kinase (3). Activation of phosphorylase kinase by calcium causes a conversion of the inactive form of phosphorylase to the active form, resulting in increased glycogenolysis. Alternatively, fasting is known to deplete liver glycogen stores via glucagon action (2), but does not influence muscle glycogen reserves. Skeletal muscle is not responsive to glucagon action, likely because of very low levels of glucagon receptor, as reflected by negligible receptor mRNA versus 1000-fold greater expression in liver (4) and thus, low levels of glucagon receptor activity. Surprisingly,

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several studies have shown decreases in muscle and carcass glycogen levels with feed deprivation (5–7). Using cervical dislocation (CD) to collect tissues, recent literature has reported time-dependent losses in muscle glycogen with prolonged feed withdrawal (FW; (5)). However, CD involves extensive and strenuous muscle contraction, and would therefore be expected to induce muscle glycogenolysis and confound apparent responses to other factors. Thus, these previous results are questionable in the context of the known mechanisms of muscle glycogen regulation as documented in mammalian species (2).

The objective of this study was to more accurately evaluate the effects of both feed deprivation and refeeding on avian muscle and liver glycogen concentrations, by using methodology that precludes artifactual glycogen breakdown. In addition, concentrations of circulating glucose, glucagon, and insulin were related to changes in muscle and liver glycogen during various nutritive states, to provide a more comprehensive profile of metabolic regulation of avian tissue glycogen. Finally, existing protocols for determination of tissue glycogen use either strong acid or alkaline hydrolysis, which have been demonstrated to induce partial and variable hydrolysis of the glycogen polymer (8,9). Therefore, a method based on cold, dilute perchloric acid extraction of tissue glycogen was established and validated for use with muscle and liver.

MATERIALS AND METHODS

Animals. Broiler chickens produced by crossing Avian Farms male line males with Avian Farms female line females (Avian × Avian) were reared on litter at the Pennsylvania State University Poultry Education and Research Center until 5 wk of age. All birds were maintained under a 23:1 hr (light:dark) cycle. For a study involving FW and refeeding (Experiment 2), male birds were fed a commercial broiler starter (20.50% crude protein, 2,300 kcal ME/kg, 5.5% fat, 3.5% fiber; Pennfield Co., Lancaster, PA) for 3 wk, followed by a commercial broiler grower (19% crude protein, 2,300 kcal ME/kg, 5% fat, 3% fiber) for an additional 3 wk. For the other studies, female birds were fed a commercial broiler starter only (20.50% crude protein, 2,300 kcal ME/kg). At 5 wk of age, the birds were moved to batteries and allowed to acclimate for 1 wk, before the initiation of treatments. All animal procedures were approved by the Pennsylvania State University Institutional Animal Care and Use Committee.

Experiment 1: Method of Tissue Collection. To evaluate the effects of CD versus other methods of tissue collection on muscle glycogen levels, 6-wk-old female broiler chickens were killed by one of four methods (10 birds/treatment): 1) barbiturate overdose (60 mg/kg body weight sodium pentobarbital administered by rapid iv bolus injection) followed by rapid freezing (<20 s from barbiturate injection to freezing) of tissue (*pectoralis superficialis*) in liquid nitrogen (OD 0); 2) barbiturate overdose followed by freezing of tissue in liquid nitrogen 3 min after injection (OD 3); 3) barbiturate overdose followed by freezing of tissue in liquid nitrogen 10 min after injection (OD 10); and 4) CD followed by freezing of tissue in liquid nitrogen 3 min after initiation of CD. Tissues not immediately frozen remained in the bird until the end of the holding period.

Experiment 2: Feed Withdrawal/Refeeding. Tissue samples were collected by using barbiturate overdose followed by immediate freezing in liquid nitrogen to accurately determine the changes in avian muscle and liver glycogen levels that occur with FW and refeeding. Muscle (*pectoralis superficialis*) and liver samples were collected from groups of 6-wk-old male broiler chickens before and after a 24-hr FW, at the end of 12-hr of refeeding, and from continuously fed controls at each interval (10 birds/treatment). To associate changes in glycogen levels with insulin, glucagon, and glucose concentrations,

blood samples were taken at the initiation and end of a 24-hr feed-deprivation period, at various times after refeeding (15 min, 1 hr, 6 hr, and 12 hr), and from continuously fed controls at each interval. Samples were collected into 1.5-ml microfuge tubes (VWR Scientific) containing 15 μl leupeptin (0.0119 g leupeptin/ml phosphate-buffered saline; Boehringer Mannheim) and 23 μl of 10% ethylenediaminetetraacetic acid, centrifuged in a Beckman microfuge (Model B; Beckman Instruments) for 2 min, and plasma stored at −80°C.

Experiment 3: Prolonged Feed Withdrawal. To determine the effect of an extended feed-deprivation period on muscle glycogen levels, 6-wk-old female broiler chickens were deprived of feed for 0, 12, 24, 48, or 72 hr. Birds were killed by barbiturate overdose and samples immediately frozen in liquid nitrogen. All samples were stored at −80°C until glycogen content was determined.

Plasma and Tissue Assays. A homologous radioimmunoassay was used for plasma insulin determinations as described previously (10). Only one assay was required to determine insulin concentrations, with an intraassay coefficient of variation of 3.4%. Plasma glucagon was determined as previously described (11), by using reagents purchased from Linco Research Immunoassay (St. Charles, MO). One assay was required to determine glucagon concentrations, with an intraassay coefficient of variation of 2.2%. Plasma glucose concentrations were determined with a Beckman Glucose Analyzer 2 (Beckman Instruments, Inc.).

For both liver and muscle glycogen determinations, all samples and standards were analyzed in duplicate. A glycogen assay for determination of tissue glycogen, which would avoid degradation of the glycogen polymer, was adapted from Dalrymple and Hamm (8) and validated. Tissue samples were homogenized in 0.6 mol/liter perchloric acid by using a Tekmar SDT1810 Tissumizer with SDT100EN shaft (Tekmar, Cincinnati, OH). One aliquot of each homogenate and aliquots of glycogen standard solutions made from purified glycogen (Sigma) were hydrolyzed with 0.5 mg amyloglucosidase (EC 3.2.1.3, Sigma, Cat.# A 7420) in 1 ml of 0.2 mol/liter acetate buffer, pH 4.8. Potassium hydroxide (20 μl of 5.4 mol/liter) was added and hydrolysates were allowed to incubate at 40°C for 2 hr. As a control for determination of free glucose, 1 ml of 0.2 mol/liter acetate buffer, pH 4.8, and 25 μl of 4.32 mol/liter potassium hydroxide was added to a second aliquot of hydrolysate and incubated for 2 hr at 40°C in the absence of amyloglucosidase. Cold 3 mol/liter perchloric acid (100 μl) was then added to precipitate protein. The precipitate was allowed to settle for 10 min at 0°C, followed by centrifugation at 1,000 × *g* for 10 min at 4°C. The supernatant was decanted and held at 4°C. A glucose oxidase method (Sigma, Cat.# 510-A) was used to determine free glucose in a 0.5-ml aliquot of supernatant. To verify that the protocol of cold perchloric acid extraction and enzymatic (amyloglucosidase) hydrolysis was valid for determination of tissue glycogen, serial dilutions of muscle extracts and recovery of purified glycogen standard (Sigma, Cat.# G8876) added to a muscle extract were assessed.

Statistical Analyses. All results are expressed as least square means ± SEM. For plasma hormone and metabolite concentrations, data were analyzed by a repeated measures ANOVA executed by using the General Linear Models procedure of Statistical Analysis Systems (SAS Version 6.08, SAS Institute, Cary, NC). Differences between treated groups and the control were determined by using Boneferroni's test (12). For evaluation of tissue glycogen differences, data were analyzed by a one-way ANOVA by using the General Linear Models procedure of SAS and means separated by Boneferroni's test. Where no significant differences were detected among the continuously fed controls for each interval of the FW and refeeding scheme, a pooled control group was used for comparison between the control and treated groups.

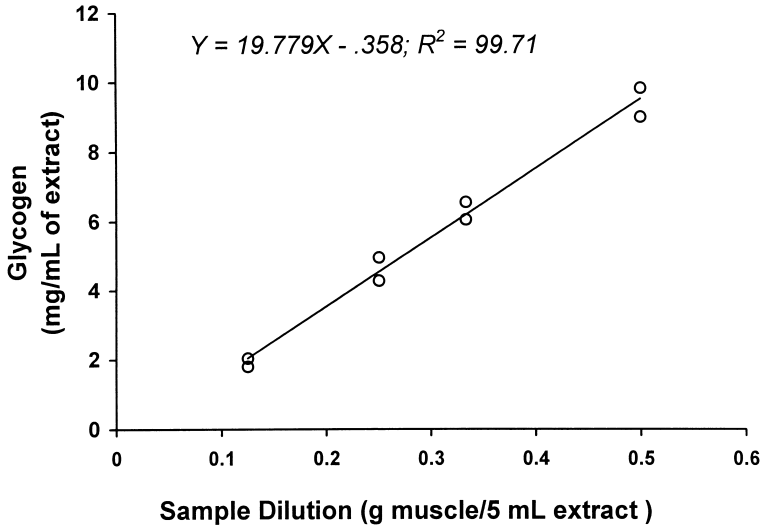

Figure 1. Linear response in glycogen concentration to serial dilutions of skeletal muscle extract over the range of 0.125 to 0.5 g tissue/5 ml perchloric acid extract by using an assay method based on perchloric acid (0.6 mol/liter) extraction/homogenization followed by amyloglucosidase hydrolysis (all samples were assayed in duplicate).

RESULTS

To verify the use of cold perchloric acid extraction and enzymatic (amyloglucosidase) hydrolysis for chicken glycogen determination, serial dilutions of muscle extracts and recovery of purified glycogen standard added to a muscle extract were assessed. As the quantity of muscle extract increased, glycogen content increased in a linear fashion (Figure 1). The percent recovery for each amount of purified glycogen added from 0.5–8 mg averaged approximately 117% (Table 1). Glycogen concentrations were not corrected for free glucose because free glucose measured in each sample was negligible and below the lowest standard curve concentration (2 mg/g).

Killing birds by CD, in the absence of feed deprivation, decreased muscle glycogen by 23% (Figure 2) as compared with control levels in tissues rapidly collected from birds killed by barbiturate overdose (OD 0). No significant differences were found between control muscle glycogen levels and glycogen levels in muscles collected 3–10 min postmortem in barbiturate overdosed birds (OD 3 and OD 10; $P < 0.05$).

TABLE 1. GLYCOGEN CONTENT AND PERCENT (%) RECOVERY FOR INCREASING AMOUNTS OF ADDED PURIFIED GLYCOGEN[a]

Glycogen Added (mg) Recovery	Glycogen Content (mg/ml)	%
0	0.88 ± 0.40	—
0.5	1.47 ± 0.40	117%
1	2.12 ± 0.40	124%
2	3.21 ± 0.40	116%
3	4.47 ± 0.40	120%
4	5.95 ± 0.40	127%
6	7.12 ± 0.40	104%
8	10.37 ± 0.40	112%

[a] All samples were assayed in duplicate. Purified glycogen (Sigma) was added to 0.2 g/ml muscle extract and glycogen amount determined by using an assay method based on cold perchloric acid extraction/homogenization followed by amyloglucosidase hydrolysis. Glycogen content is expressed at least square means ± SEM.

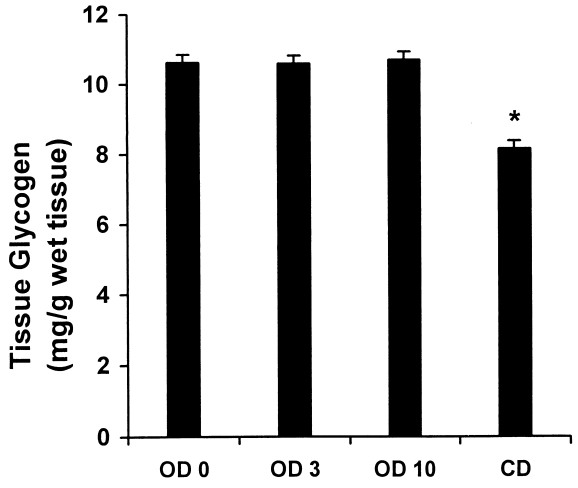

Figure 2. Changes in muscle glycogen with method of tissue collection. Samples were collected from 6-wk-old female broiler chickens by one of four methods (10 birds/treatment): 1) Barbiturate overdose followed by rapid freezing (<20 s) of tissue *(pectoralis superficialis*) in liquid nitrogen (OD 0); 2) Barbiturate overdose followed by freezing of tissue in liquid nitrogen 3 min after injection (OD 3); 3) Barbiturate overdose followed by freezing of tissue in liquid nitrogen 10 min after injection (OD 10); or 4) Cervical dislocation (CD) followed by freezing of tissue in liquid nitrogen 3 min after initiation of CD. Data are expressed as least square means. An asterisk indicates a significant difference from the OD 0 value ($P < 0.05$).

Plasma glucose was decreased, whereas glucagon was elevated after 24-h of withdrawal when compared with control concentrations ($P < 0.05$; Table 2). By using barbiturate overdose to kill the animals, a method that minimizes both stress and muscle contraction, substantially higher ante-mortem muscle glycogen concentrations were observed (Figure 3) than had been reported previously in the chicken (5). After the 24-hr FW, liver glycogen decreased by 77% compared with control levels ($P < 0.05$), whereas muscle glycogen decreased by only 13% ($P < 0.05$). Feed deprivation followed by refeeding resulted in increased insulin and glucose levels ($P < 0.05$) when compared with control levels (Table 2). After refeeding, liver glycogen increased 380% over control values, whereas muscle glycogen did not change significantly from the control value ($P < 0.05$; Figure 3).

With prolonged FW, muscle glycogen was transiently reduced by 13% ($P < 0.05$) at 24 hr of FW, but had returned to control levels by 48 hr of withdrawal, and remained at that concentration even at 72 hr of feed deprivation (Figure 4).

TABLE 2. CHANGES IN PLASMA INSULIN, GLUCAGON, AND GLUCOSE CONCENTRATIONS WITH FEED WITHDRAWAL AND REFEEDING IN 6-WK OLD MALE BROILER CHICKENS[A]

Time	Insulin (ng/ml)	Glucagon (pg/ml)	Glucose (mg/dl)
Feed Withdrawal			
0	2.7 ± 0.45	176.2 ± 12.68	217 ± 4.3
24 hr	2.7 ± 0.45	248.1 ± 12.68[a]	171 ± 4.3[a]
Refeeding			
15 min	7.2 ± 0.45[a]	137.0 ± 12.68	267 ± 4.3[a]
1 hr	5.1 ± 0.45[a]	136.3 ± 12.68	246 ± 4.3[a]
6 hr	4.7 ± 0.45[a]	139.9 ± 12.68	234 ± 4.3[a]
12 hr	3.1 ± 0.45	158.5 ± 12.68	246 ± 4.3

[a] Data are expressed as least square means ± SEM. Values are means of repeated measures on the same animals ($n = 10$). An asterisk indicates a significant difference from the ad libitum-fed group ($P < 0.05$).

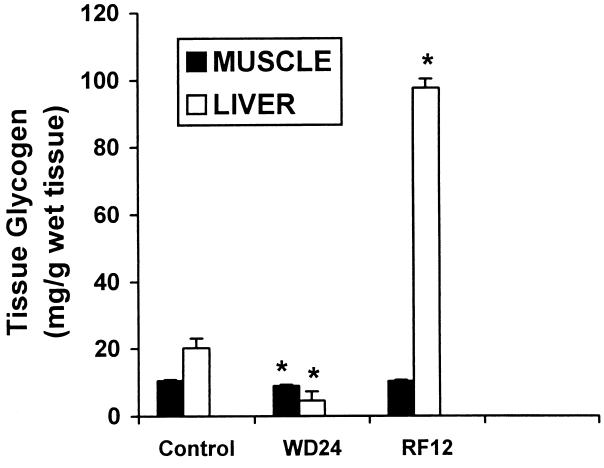

Figure 3. Changes in muscle and liver glycogen in 6-wk-old male broiler chickens with a 24-hr FW (WD24) and a 12-hr refeeding period (RF12). Data are expressed as least square means (control: $n = 30$; WD24 and RF12: $n = 10$). An asterisk indicates a significant difference from the ad libitum-fed control value within a tissue type ($P < 0.05$).

DISCUSSION

In the course of tissue glycogen determinations, the use of strong alkali, strong acid, or high temperatures may degrade tissue glycogen (9) or cause incomplete glycogen precipitation (8) during homogenization. Higher levels of glycogen attributed to protein-bound glycogen and ethanol soluble carbohydrate are measured with perchloric acid homogenization, when compared with the use of potassium hydroxide-ethanol homogenization (8). Use of amyloglucosidase has been shown to yield equivalent glycogen amounts as hydrolysis with phosphorylase and glycogen debranching complex [two domains: 4-α-glucanotransferase (2.4.1.25) and amylo-1,6-glucosidase (3.2.1.33)] the enzyme system used naturally by the cell for glycogenolysis (13). In this study, cold perchloric acid

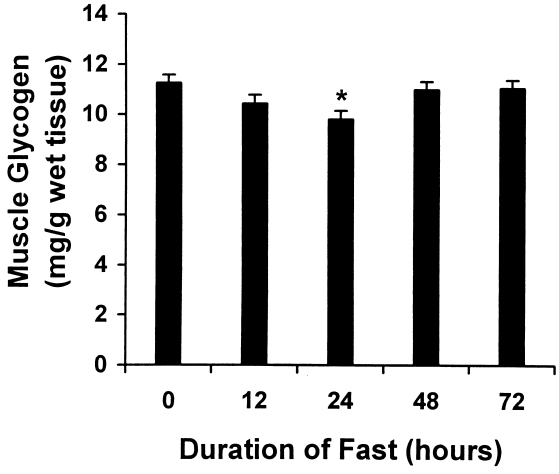

Figure 4. Effect of prolonged FW on muscle glycogen levels. Feed was withdrawn from 6-wk-old female broiler chickens for 0, 12, 24, 48, or 72 hr (8 birds/withdrawal period). Data are expressed as least square means. An asterisk indicates a significant difference from the ad libitum-fed control value ($P < 0.05$).

extraction and enzymatic hydrolysis for determination of tissue glycogen were validated based on serial dilution of tissue extracts and glycogen recovery data.

Because the tissues in both the OD 3 and CD groups remained in the animal for the same 3-min period, the results of the method of tissue collection experiment indicate that the decline in muscle glycogen from control values in CD birds does not represent postmortem decomposition, but rather effects associated with CD (e.g., stress and reflex muscle contraction). By using CD to collect samples, Kotula and Wang (5) found decreases in muscle glycogen in birds subjected to prolonged FW. However, the current understanding of the hormonal regulation of glycogen metabolism in mammalian species, in which both catecholamine release during stress and calcium release into the sarcoplasm during muscle contraction can activate phosphorylase kinase, and thereby stimulate glycogen breakdown (2), are consistent with the findings in avian tissues in the present study.

The elevated plasma glucagon levels during FW were associated with a substantial decrease in liver glycogen, but not muscle glycogen, which is consistent with the fact that there is minimal glucagon receptor expressed in muscle (4). The differential response to feed deprivation in muscle versus liver has been widely documented in the rat (14–17), although the magnitude of muscle glycogen depletion is greater than that reported in this study. Additionally, the amount of depletion in rat muscle glycogen varies greatly by fiber type (17,18). Therefore, the difference in magnitude of decrease in rat versus chicken muscle glycogen in response to feed deprivation may relate to the unique composition of chicken pectoralis muscle, which consists almost entirely of αW fibers.

In mammals, insulin is a known stimulator of glycogen synthase (EC 2.4.11), the rate-limiting enzyme in glycogen synthesis (19,20); therefore, the increase in plasma insulin in the present studies is likely a key hormonal stimulus for the observed elevated tissue glycogen concentrations. After refeeding, liver glycogen increased 380% over control values, whereas muscle glycogen did not change significantly from the control value. The supercompensation of liver glycogen after refeeding is well established in mammals (14–17,21,22), which again illustrates the similarities between avian and mammalian glycogen regulation. It is not known whether an increase in muscle glycogen would have been detected if an earlier sampling interval had been used. For example, Holness et al. (17) found an elevation in glycogen ranging from 38% to 66% over control values in various rat muscle fiber types during the first 8 hr of refeeding, after which glycogen levels returned to control concentrations. The small decrease in muscle glycogen with feed deprivation may also have precluded any supercompensation effect, because the magnitude of glycogen depletion has been shown to influence the rate of muscle glycogen resynthesis in humans after exercise (23). Furthermore, the activity of glycogen synthase has been demonstrated to vary inversely with muscle glycogen concentrations (24–26). Perhaps in the chicken, as with mammalian species, significant depletion of muscle glycogen is required to enable significant supercompensation after refeeding.

The results of the current study indicate that muscle glycogen is not very responsive to nutritive status, unlike the results of previous work in the chicken. Kotula and Wang (5) found decreases in muscle glycogen in birds subjected to prolonged FW times, by using CD to kill the birds. However, because broiler chickens become hyperirritable as FW progresses, more struggle and muscle contraction may occur upon CD as the period of feed deprivation before killing is increased. Also, enhanced epinephrine release is likely to occur, which is known to stimulate muscle glycogenolysis (27). Therefore, the time-related changes in muscle glycogen observed by Kotula and Wang (5) may in part reflect proportionately greater stress and muscle contraction at the time of sample collection in birds subjected to prolonged FW. Additionally, the effects of prolonged FW on reducing

blood glucose concentrations may interact with muscle contraction to differentially reduce muscle glycogen upon CD. In the human, maintenance of blood glucose helps to preserve muscle glycogen during regimens of endurance training (28). In the present study, fasting-induced hypoglycemia was observed, which may have exacerbated the response of muscle to stress and involuntary muscle contraction upon CD. As fasting progresses, the degree of hypoglycemia increases, and, therefore, the extent of muscle glycogenolysis upon CD may be more severe. This may explain the apparent greater losses in muscle glycogen in birds subjected to increased FW times, as reported by Kotula and Wang (5).

In conclusion, the present study verifies that feed deprivation in the chicken does result in greatly decreased liver glycogen, and that liver glycogen has enormous potential for supercompensation after refeeding. However, feed deprivation and refeeding does not have a major, sustained effect on avian muscle glycogen levels, even after prolonged withdrawal periods of 72 hr. This illustrates the similarities between avian and mammalian muscle glycogen regulation, in contrast with previous reports (5,7) that erroneously concluded that direct provocation of muscle glycogenolysis by feed deprivation occurs in the bird. Muscle glycogen is resilient to the metabolic changes that occur during feed deprivation and refeeding, in contrast with the labile nature of liver glycogen.

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